Intracranial hemorrhage and extensive cerebral venous thrombosis associated with ulcerative colitis

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CASE PRESENTATION

A middle-age man with history of ulcerative colitis (UC) maintained on certolizumab and 6-mercaptopurine in addition to prednisone for a recent flare presented with altered mental status and severe headache of 6 h duration. According to his family, he had been experiencing mild intermittent headaches for the previous four weeks without any fevers, chills, blurred vision or neck stiffness. He had no history of trauma or ill contacts. Neurological examination revealed an attentive male with fluent but nonsensical speech. Motor and sensory examinations were nonfocal. A computed tomography scan of the brain showed acute hemorrhage in left temporal lobe with surrounding vasogenic edema and mass effect on the underlying brain parenchyma and left lateral ventricle. A magnetic resonance venogram revealed absence of signal within the left vein of Labbe, left transverse and sigmoid sinus, and left internal jugular vein consistent with extensive cerebral venous thrombosis (CVT) (Figures 1 and 2). Anticoagulation was initiated. His mental status subsequently deteriorated and repeat brain computed tomography scan showed worsening hemorrhage with midline shift and

uncal herniation. The patient underwent emergent decompressive left hemicraniectomy. Anticoagulation was restarted postoperatively. The patient experienced a highly favourable recovery and was able to follow simple commands and move all extremities against gravity before his discharge from the hospital.

DISCUSSION

CVT is an uncommon disorder, with an annual incidence of three to four cases per million. Risk factors for CVT include thrombophilias, malignancies, hematological disorders, sinus infections, vasculitides, traumatic head injuries and inflammatory bowel diseases. Thrombosis of cerebral veins result in increased pressure in the venous system, which ultimately results in decreased cerebral perfusion, ischemic injury, disruption of the blood-brain barrier and vasogenic edema. Occasionally, this increased pressure can lead to venous and capillary rupture causing parenchymal hemorrhage. Clinical presentation of this entity is highly variable and can include headaches, seizures, encephalopathy or focal neurological signs. Diagnosis can be delayed for the same reason and, therefore, requires high clinical suspicion. Magnetic resonance venography is the most sensitive test for detection of CVT. Systemic anticoagulation is considered to be the primary

Figure 1) Magnetic resonance imaging. Axial T2 image showing heterogeneous collection at the left temporal lobe consistent with a large intraparenchymal hemorrhage containing both subacute and acute blood. Moderate surrounding vasogenic edema (arrows)

Figure 2) Magnetic resonance venogram image showing abrupt loss of flow related signal at the proximal left transverse sinus continuing distally to involve the left sigmoid sinus, vein of Labbe and jugular vein (arrows)

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therapy for CVT even in individuals presenting with hemorrhagic infarcts (1,2). In patients who do not respond to systemic anticoagulation or those presenting with severe neurological symptoms, catheter-directed thrombolytic therapy with or without thrombectomy may be considered (3). Herniation due to unilateral mass effect is a major cause of mortality and requires decompressive surgery. The majority of patients with CVT experience either complete or partial recovery, and recurrence is rare. In a meta-analysis of 1180 patients with CVT (4), the mean 30-day mortality was 5.6%.

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